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Case study

Combined motor disturbances following severe traumatic brain injury: an integrative long-term treatment approach

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Patients surviving severe traumatic brain injury (TBI) often suffer from residual impairments in motor control, communication skills, cognition and social behaviour. These distinctly hamper their capability to return to their 'pre-trauma' activity. Comprehensive and integrated rehabilitation programmes initiate, during the acute phase, a prolonged treatment process which starts at the most sophisticated medical systems. There is no clear end point for the treatment of these patients, since the recovery process and the rehabilitation activity may continue for years, even after patients return home to live with their families. The inherent inability to make a firm early prediction regarding outcome of patients and the late appearance of additional symptoms stress the need for a comprehensive close long-term follow-up. The following presentation concerns the description of the treatment strategy and long-term improvement of a 22-year-old male who suffered from very severe TBI. On admission to the emergency room, he was in the decerebrated position and his Glasgow Coma Scale (GCS) was at the lowest (3). The focus of this presentation is on the recovery of motor function. The initial motor disabilities included weakness in all four limbs, in particular left hemiplegia, and right hemiparesis with severe bilateral ataxic elements and a marked tremor of the right arm. Range of motion was limited in hips, and he suffered from stiff trunk and neck. Goals of physiotherapy were directed towards improving range of motion (ROM) and active movement. Casting, use of orthoses, biofeedback, hydrotherapy, hippotherapy, medication and nerve blocks for reducing spasticity were timely applied during the process. The motor improvement in this very severe TBI patient who is now over 3 years post-injury still continues and has a functional meaning. He has succeeded in being able to stand up by himself from a chair and is able to walk unaided and without orthoses for very short distances—up to five steps. He is able to drink soup without assistance and play a few notes on the piano. Marked cognitive improvement occurred as well. It is concluded that motor improvement may be evident over long periods of time and various timely interventions may assist in the process.

Introduction

Traumatic brain injuries (TBI) are known to cause a diversity of disorders involving motor, sensory, cognitive and behavioural dysfunction [1]. The motor disabilities following TBI can be diverse, since the neural damage post-TBI can be distributed throughout many areas of the central nervous system [2]. Motor performance can be affected by damage to various loci in the nervous system, such as cortical and sub-cortical areas as well as brain stem. The functional consequence may be related

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to a reduction in muscle power (paresis), to sensory disturbances interrupting feedback and feed forward mechanisms, tonus disorder (spasticity), co-ordination, and motor control disorganization [2].

The overlap between the various disciplines involved in the rehabilitation process is well documented in the literature. However, the time frames under which they operate are less well documented. The current case presentation focuses on the motor domain and highlights the need for long-term integrative treatment using timely applied modes of therapy in order to maximize outcome.

Case presentation

A 22-year-old male was involved in a road traffic accident resulting in severe TBI. On admission to the emergency room he was in the decerebrated position and his GCS [3] was the lowest, i.e. 3.

Ten weeks after the injury he was transferred to the coma unit of a large rehabilitation centre. At this time, he was still unconscious with no response to pain, he was breathing spontaneously and his eyes were open, with pupil reaction being equal and responsive to light. Physical examination revealed no active movements but full passive range of motion was available. High tonus was present in the right arm and left wrist and fingers with increased tendon reflexes. No abnormal tone was detected in the lower limbs and there were no discernible tendon reflexes. Tracheotomy was still *in situ* and he was fed via a gastrostomy tube.

Since the earliest days post-trauma, he had demonstrated tremor of the head and right shoulder girdle. This was reduced pharmacologically with anti-epileptic and anti-spastic drugs (Sod. Valporate and Clonazepan). Recurrent EEG recording showed no signs of epileptic foci.

The first signs of communication appeared at ~4 months post-injury, obeying a command in the form of eye opening and closure in response to yes/no questioning. A further 2 months later, following removal of the tracheotomy and lightening of the unconscious state, communication improved with increased gestures and the use of a communication board.

Motor changes also occurred at this time. The excessive hyperextension of the head and neck was reduced to almost nil, and spontaneous movements were present in the elbow, wrist and fingers of the right arm. Extensor tone developed in the left upper limb and flexor tone in the right upper and lower limbs. Functional improvement enabled him to sit in a wheelchair for short periods of time.

Eight months post-injury, he was in a fully conscious state and was transferred to an active rehabilitation ward. His communication abilities by now included emission of sounds and indistinct words. Motor disabilities were described as weakness in all four limbs, in particular left hemiplegia, and right hemiparesis with a severe bilateral ataxic element. He also had a marked tremor of the right arm. Range of motion was limited in hips, knees and ankles of both sides, but X-rays did not reveal any heterotopic new bone formation. The excessive tone was pharmacologically treated by Sodium Dantrolene and by applying Phenol blocks to appropriate muscle groups. Inhibitory casting and daily standing on the tilt table (30 minutes per day) were also applied.

During the following 6 months, he continued to make slow but steady progress. Full passive range of motion was gained and maintained in both upper and lower limbs. A resting cast splint was built in the functional position for the left wrist,

allowing him to have individualized finger movements, whilst controlling the wrist in extension.

Unsupported sitting balance was achieved, as was standing balance with support of his right hand. He was able to 'walk' 3 metres with the use of a long leg brace and one person for support. The tremor in the right arm was better controlled, although the limb remained essentially non-functional. In the following 3 months, he attained a gait pattern without the leg brace and was now able to ambulate up to 10 metres with the assistance of one person. No suitable gait aid was found to be of use due to the strong truncal ataxia. At about this time, biofeedback was also used for improving fine finger movements. Cognitive function continued to improve parallel to the gains in motor function.

Twenty-two months after injury, he was discharged home, but continued therapy at the Day Care TBI Unit for 5 days a week. In addition to physiotherapy, he was treated for his motor disturbances by hydrotherapy, occupational therapy and hippotherapy.

Treatment aims at this stage were more focused towards three specific areas:

(1) The right hand was the better one relating to movement, but it remained non-functional due to tremor. A test evaluation by triaxial accelerometry revealed that there was no resting tremor; however, every active movement of his right side triggered tremor of the pectoralis major, triceps and biceps muscles, both against and with gravity. The tremor frequency was between 2–4 Hz.
(2) The left hand, that began as the hemiplegic hand, gained gross motor function but was still exhibiting high tone which interfered with any type of isolated movement.
(3) He was still exhibiting severe balance problems in both sitting and standing positions with severe increased tone in the neck; the increased tone was asymmetrical, showing torticollis to the left.

It was, therefore, decided to address these three major problems step by step, First, a nerve block, using Botulism toxin to the flexors of the wrist and the fingers of the left hand, was applied. The injections were followed 1 week later by a removable light cast, allowing subsequent stretches throughout. The Abductor and Opponens Pollicis showed the greatest reduction in tone, and this allowed a better grip function resulting in the use of regular utensils. The patient was, thus, now able to feed himself and to write with a pen with his left hand. Subsequent Botulinum toxin injections produced a marked increase in dorsiflexion of the left wrist, allowing 4-point kneeling to be achieved and, thus, better weight bearing on the left upper limb.

The 2nd step was injection of Botulinum toxin to the left Sternocleidomastoid and Trapezius muscles. Again, dramatic results were achieved within 7 days and static standing balance increased to >30 seconds, thereby significantly improving his gait pattern.

In the 3rd step, the authors tried to reduce the tremor of the right arm, as this had not previously responded to either physical or pharmacological forms of treatment. It was decided to use Botulinum toxin to the more distal muscle groups. The injections were given on two separate occasions with a 2-week interval between; first, to the more proximal muscles—Biceps and Triceps, and then to the more distal groups—the Extensor Carpi muscles.

After the first injection, a reduction in the tremor appeared, but without any functional improvement. However, after the second injection, a further meaningful reduction in the tremor was evident, enabling the patient to drink soup with his right hand and even to play few notes on the piano.

Approximately 3 years after the injury, the patient is now able to stand up alone from a chair and walk, without orthoses, unaided for very short distances—up to five steps. With a regular walking frame, he is now able to walk up to 50 metres. He is also able to go up and down stairs with the use of the handrail and one assistant.

Discussion

Motor disturbances post-TBI involve many and various components of the motor control system and, as such, motor recovery may be gradual and prolonged [4]. Some functional motor improvements may only be achieved several years post-injury. This is particularly obvious in the more severely affected TBI patients.

Motor recovery usually precedes cognitive and behavioural improvements. Higher cortical function would seem to be particularly influenced by motor recovery. In the Stroke population, major motor improvements are usually achieved during the first 3 months post-insult [5–7]. Although the steepest gradient of functional improvements in TBI patients is usually seen in the immediate post-coma phase [8], there are exceptions to this common rule. This is true, according to the authors' experience, for the recovery of fine motor control, which may happen only after 2 years or more, post-injury.

The very late motor recovery seen in this patient may be explained in several ways. It is worth mentioning that over half of TBI patients suffer from polytrauma. Even in so called 'pure' head injuries, systems like the immune system are deeply involved. It has been shown, by Wolach *et al.* [9], that there is a delayed activation of the immune system due to a humoral defect in severe TBI patients. Zasler [10] also noted functional abnormalities of the polymorphonuclear leucocytes and complement among numerous other immunological changes in patients following multi-trauma. Diffuse axonal injury is the common pathology in blunt head injuries and, as such, various combinations and varieties of functional motor deficits may be evident. An integrated well-functioning motor system needs the proper functioning of the various modules of the system, each of which may have its individual rate of recovery. For example, movements may exist in a limb, but proper function may be sometimes hindered due to spasticity or tremor. Therefore, proper function will be achieved only after controlling the abnormal phenomena or their recovery, thus leading to delayed rehabilitation. In comparing this to normal neuronal development, it is known that a child can only achieve certain motor functional activities after maturation of the central nervous system allowing him to explore the environment. For example, walking in children is attained in the 2nd year of life, even though some components like automatic gait are present at birth.

The major motor disturbances following TBI will include reduction of power production by muscles (paresis), sensory disturbances (interruption of the feedback and feed forward mechanisms), tonus disorder (spasticity), and coordination and motor control disorganization. The rational treatment protocol for these integrative disorders must be based on co-operation among medical and allied health professionals, especially in the fields of physiotherapy and occupational therapy in order to maximize residual potentials. For example, reduction of spasticity was achieved by

several means. The decision to use nerve block as a method of reducing spasticity was analysed by the multidisciplinary team. Questions like when to use nerve blocks, the timely selection of appropriate muscle groups and whether phenol or Botulinum toxin should be used, had to be answered. It was decided to first apply phenol nerve blocks to the more proximal robust muscle groups and then to use Botulinum toxin in the more fine muscle groups of the hand.

The nerve blocks were administered in combination with pharmacological therapy and dynamic physical therapy. It has been shown, by Hesse *et al.* [11], that dynamic intervention following Botulinum toxin injection may facilitate its activity and improve gains in functional achievement from the combined therapy.

The use of Botulinum toxin in this patient had three different aims:

(1) *Reducing spasticity*. To reduce spasticity, thus allowing improvement of ROM and functional activity. The most obvious indication for the use of Botulinum toxin is to reduce the activity in over active muscles. The toxin produces temporary muscle weakness by pre-synaptic inhibition of acetylcholine release at the neuromuscular junction [12]. The meticulous pre-injection assessment must involve physicians and physiotherapists in order to precisely define the over-active muscles and to predict the optimal outcome and structure therapy following injection. Motor performance may often continue to improve long after the pharmacological effect of the toxin has worn off.

(2) *Improving balance*. Balance reactions have been improved following the reduction in spasticity in this patient. The decreased muscle tones of the neck and upper trunk improved the static balance, i.e. stability while standing. Since stability is the forerunner to mobility, they gained increased stability, and helped in normalizing his gait pattern. It was the authors' impression that this effect continued long after the presumed theoretical effect of the toxin.

(3) *Reducing tremor*. As a consequence of TBI, two distinct sets of symptoms may be described, the positive and the negative. The positive features being a release phenomenon or exaggeration of activity and the negative features being a paucity of movement [13]. Botulinum toxin is a specific agent that reduces the over-activity of the muscle at the pre-synaptic level. The inhibition at this level has also been demonstrated to have a partial effect on essential tremor [14–16]. The rationale of introducing this therapy was based on the results reported by Rondot *et al.* [17], of transient suppression of forearm tremors following clinically guided intra-muscular injections of lidocaine. Many questions regarding this approach to treatment still remain: First, is there sectional activity in essential tremor and what is the difference between essential tremor and focal dystonia? Secondly, how should one choose the appropriate muscle targets? Thirdly, what is the optimal dose? Fourthly, should the treatments be given in one session or more? Fifthly, what is the recommended follow-up treatment? And, finally, how should the efficacy of treatment be assessed?

In the presented patient, the aetiology of the tremor was TBI. Very few such cases have been reported in the literature [18]. The results demonstrate that maximum effectiveness on all occasions was gained only after the second injection. This concurs with the results reported by Jancovic *et al.* [16]. The muscles chosen for

injection were the distal groups, even though it was very difficult to isolate the exact source of the tremor. It has been speculated by Hallet [19] that the beneficial effect of Botulinum toxin treatment may also be mediated by a blockade of the gamma motor efferents and muscle spindle afferents.

Treatment of TBI patients is a multi-stage complex process. It starts at the place of injury, and continues preferably at a hospital provided with a neurosurgical unit and a trauma centre. Then, through various other interim institutions, rehabilitation programmes, special TBI inpatient and outpatient programmes and follow-up clinics. The process can be long and it should be noted that damage inflicted in seconds might require years of rehabilitation. The motor system, especially in the more severe TBI patients, may require long periods of time and complex handling in order to recover and enable a better functional outcome.

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